LETTERS TO THE EDITOR

Cereal fibre and rectal epithelial cell proliferation: a possible link with duodenal ulceration

EDITOR,—It is interesting that Macrae *et al* (*Gut* 1997;**41**:239–45) found that unprocessed wheat bran had a greater effect than processed bran or oat bran on the reduction of labelled cells in the top two fifths of rectal epithelial crypts. There is evidence that wheat bran may have effects on other mucosal surfaces, particularly that of the duodenum. The prevalence of duodenal ulceration is lower in India in the unrefined wheat eating areas of the Punjab than in the polished rice eating areas of the South.[1–3] Our experimental work on several animal models has shown that wheat bran is protective against peptic ulceration and that this protection is linked with its lipid content.[4 5] The evidence so far suggests that this effect may be related to a unique combination of lipids in certain cereal fibres and not to a particular type of fibre.

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1 Tovey FI. Peptic ulcer in India and Bangladesh [progress report]. *Gut* 1979;**20**:329–47.
2 Tovey FI, Jayaraj AP, Lewin MR, *et al*. Diet: Its role in in the genesis of peptic ulceration. *Dig Dis* 1989;7:309–23.
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"Golf ball liver": a cause of chronic hepatitis ?

EDITOR,—A 23 year old man was referred to the outpatient clinic in view of persistently abnormal liver function tests for five years. In 1992, he had a gastrointestinal illness manifest by abdominal pain, diarrhoea and vomiting which lasted intermittently for one month. At that time, investigations by his general practitioner revealed that his liver function tests were abnormal: alkaline phosphatase (ALP) 408 U/l, aspartate aminotransferase (AST) 383 IU/l, alanine aminotransferase (ALT) 922 U/l, γ-glutamyl transferase (GGT) 147 IU/l. Viral serology for hepatitis A and B and a Paul–Bunnell test were negative. His symptoms resolved, but his liver function tests did not return to normal.

He was referred to the medical outpatient clinic in 1997 because of the development of further symptoms: episodic abdominal pain, diarrhoea, itch, and fatigue. His urine was noted to be dark and his faeces was pale. Alcohol consumption was moderate and confined to social occasions. He had no history of recent foreign travel, substance or drug misuse. He had no contacts with jaundice and no previous blood transfusions. Liver function tests were persistently abnormal at that time: ALP 622 U/l, AST 86 IU/l, ALT 193 U/l, GGT 555 IU/l. Other investigations including full blood analysis, erythrocyte sedimentation rate, coagulation screen, urea and electrolytes, serum ferritin, serum copper, and alpha-1-antitrypsin were normal. Analysis of plasma proteins revealed albumin 45 g/l, globulin 37 g/l, total protein 82 g/l. IgG (30.3 g/l) and IgM (4.65 g/l) were raised. Titres of antinuclear antibody (IgG 1:20) and anti-smooth muscle antibody (IgG 1:40) were low. Viral serology for hepatitis A, B and C and Epstein–Barr and cytomegalovirus were negative. Abdominal ultrasound revealed an enlarged spleen, which was not palpable, and normal liver architecture.

Percutaneous liver biopsy revealed nodules separated by fibrous bands and a moderately dense mononuclear cell infiltrate within the portal tracts and fibrous bands, composed mainly of lymphocytes. The bile ducts were well preserved. There was moderately active lymphocytic piecemeal necrosis. Stains for Wilson's disease and alpha-1-antitrypsin deficiency were carried out and were negative. The appearances suggested chronic hepatitis progressing to cirrhosis.

This young man has been a very keen amateur golfer for many years and licks his golf balls during every round. All investigations as indicated above have failed to reveal a specific cause for his chronic hepatitis and cirrhosis. In view of the recent case report (*Gut* 1997;**40**:687–8) relating acute hepatitis to inadvertent ingestion of 2,4-D, it is proposed that ingestion of this substance by golfers may also lead to chronic hepatitis and even cirrhosis as in the case of this young man with no other apparent cause for his liver disease. The results of abstention from licking his golf balls are eagerly anticipated.

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Reply

EDITOR,—We were interested to read the letter by Johnston *et al*, further to our report in the July issue, which appears to document a case of chronic active hepatitis possibly caused by the patient's habit of licking his golf balls. It appears that the habit of golf ball licking is certainly not confined to Ireland and appears, at least anecdotally, to be a relatively common practice. Given this widespread practice and the now documented ability of 2,4-D to cause an acute hepatitis, it is certainly within the realms of possibility that a chronic hepatitis may also result from chronic exposure to 2,4-D.

However, the case reported by Johnston *et al* requires some clarification. It would be helpful to know whether any eosinophils were seen on the liver biopsy sample, and if not readily seen on straightforward stains then possibly monoclonal markers such as EG1 or EG2 could be used. This is of importance as if chronic golf ball liver exists then it raises the question of whether it is a toxic phenomenon or a chronic allergic/hypersensitivity process.

It would also be of interest to know whether the authors documented that 2,4-D was actually being spread on the patient's golf course. How long had the patient been licking his golf balls prior to the intial illness in 1992, and had the chemicals used on the patient's golf course changed in or around this time. Was there any temporal relation between the patient's symptoms and the times of heaviest concentration of 2,4-D on the golf course or the frequency of this patient's golfing excursions? We await with interest any further developments.

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Mesalazine as a maintenance treatment in Crohn's disease

EDITOR,—We read with interest the occasional viewpoint by Sahmoud and Mary (*Gut* 1997;**40**:284–5). As is too often the case in such reviews, the authors have failed to identify all of the relevant literature pertinent to the topic: the maintenance of remission or prevention of recurrence of Crohn's disease. For example, with regard to azathioprine, rather than two trials of treatment, actually six trials (including the NCCDS Parts I and II) have been published. The results were summarised recently in a meta-analysis.[1] This systematic review concluded that the common odds ratio for maintaining remission for azathioprine treated patients was 2.3 (95 confidence interval (CI) 1.8 to 2.9) and that there was a relation between the cumulative dose of azathioprine prescribed and the response. Azathioprine should be considered an option for maintenance treatment.

The authors have also selectively reviewed the literature with regard to mesalazine. Turning to the postoperative population, a large North American trial[2] which identified a benefit for mesalazine for prevention of recurrence has been ignored, as well as trials which used endoscopic recurrence as a surrogate end point.[3] A second systematic review, published in the same year as that of Messori *et al* reached similar conclusions and has been updated recently.[4] Patients who received mesalazine were more likely to remain in remission compared with those on placebo. The relative risk of relapse was 0.63 (95% CI 0.50 to 0.79) for patients receiving mesalazine as compared with placebo.

We agree with the authors that additional studies are required and that differences in patient selection provides a likely explanation for differences in response. Identification of patients at higher risk of relapse could result in more cost effective treatment. Results have been conflicting with regard to clinical attributes of disease activity. While recognising that an analysis based on individual patient data might provide additional insight, the lack of consistent biological markers for high risk of recurrence (for example, permeability studies) in previous studies might make the approach irrelevant.

The issue as to whether or not to initiate maintenance treatment with either mesala-

zine or azathioprine remains a matter for discussion between patient and physician.

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1 Pearson DC, May GR, Fick GH, *et al.* Azathiprine and 6-mercaptopurine in Crohn's disease. A meta-analysis. *Ann Intern Med* 1995;**123**:132–42.
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Emergency admission to hospital for colitis due to inflammatory bowel disease

EDITOR,—We were interested to read the study by Evans *et al* (*Gut* 1997;**40**:619–22) as we have recently reported a prospective study of colitis drawing similar conclusions.[1] Our study, however, showed a much stronger association. Forty five of 62 (72.4%) new cases of colitis were taking non-steroidal anti-inflammatory drugs (NSAIDs) or salicylates compared with 38 (7%) of 513 of a control population sample (odds ratio 33.1, 95% confidence interval 17.31 to 63). This difference between the two papers is almost certainly owing to the fact that our group of colitics comprised a much broader spectrum of disease than those reported by Evans *et al*, who were clearly only studying patients with colitis in relapse.

In addition, we enquired about the usage of over-the-counter NSAIDs and salicylates which was not possible in the study design of Evans *et al*. We would maintain that exposure misclassification could easily have occurred if over-the-counter usage of these compounds was not considered.

The incident cases of Evans *et al* were de novo cases of colitis but it is not apparent why they do not regard NSAIDs as causal in these cases, as they acknowledge that these drugs have been reported as causing de novo colitis in the introductory paragraph of their paper. In our study, which was exclusively of de novo colitis, many cases have recovered completely following withdrawal of NSAIDs and salicylates suggesting a cause/effect relationship. In these patients histological differentiation from ulcerative colitis "due to inflammatory bowel disease" was not possible in the majority of cases. However, in a minority (30%) subsequent independent histological assessment has revealed some of the criteria of NSAID colitis.[2] These criteria of NSAID colitis had not been established at the time of the study (1989–93) by Evans *et al*. Thus the interpretation of their cases as Crohn's or ulcerative colitis according to the criteria of Lennard-Jones[3] may not be entirely valid.

In our opinion future epidemiological studies of the possible association of colitis and drugs need standardised histopathological review, preferably by a pathologist blinded to the drug history of the patient, which must be stringently determined by the referring clinician. It is probable that many patients classified as having "colitis due to inflammatory bowel disease" will in fact be suffering from colitis induced by NSAIDs. These drugs may be some of the most important environmental factors in the pathogenesis of colitis.

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1 Gleeson MH, Hardman JV, Clinton C. Colitis, non-steroidal anti-inflammatory drugs (NSAID's) & salicylates — a strong association [abstract]. *Gut* 1996;**39**(suppl 3):A243.
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Reply

EDITOR,—Gleeson and Warren agree with the conclusions of our recent paper, that exposure to NSAIDs is associated with colitis. Where they disagree is on the strength of the association between exposure and colitis. Unfortunately, the abstract of the study they cite contains insufficient data to make a meaningful comparison between the two studies.[1] However, one possible reason for the difference is that their control group (513 attendees at a minor injuries clinic), recalled less prior NSAID exposure than their cases. Cases (who are ill) often recall past exposure more than controls (who are not ill). Recall bias is a common and difficult problem in epidemiology. The population based dispensed prescribing data we used as our measure of exposure did not suffer from this bias.

We agree that the NSAIDs aspirin and ibuprofen which are available in low dose over-the-counter formulas, might have resulted in some exposure misclassification, but we judge this to be a small effect when compared with recall bias.

Gleeson and Warren suggest that the difference between the two studies can be explained by differences in the spectrum of disease studied. In terms of severity, this might be true. Our cases were all severe enough to warrant hospitalisation. It is possible that milder colitis might be more strongly associated with exposure to NSAIDs. Gleeson and Warren go on to suggest that NSAID colitis might be a more specific condition. Nearly all of our cases had firm diagnoses of ulcerative colitis or Crohn's colitis. It is possible that a specific NSAID induced, less severe form of colitis exists. We did not study this.

In our study incident colitis was quite strongly associated with current NSAID exposure (odds ratio (OR) 2.96) whereas non-incident colitis was not (OR 1.16). Causality is one possible explanation of an association. Our study was carried out to clarify the "signal" of possible NSAID associated colitis suggested by case reports. It seems illogical to suggest that we should then use case report evidence to strengthen our causality inference. Case reports provide a low standard of evidence of causality. They are anecdotes and the plural of anecdote is not data.

Arguments for a causal association depend upon the strength of the association, presence of biological gradient (dose response), consistency of the findings (lots of studies showing the same), and biological plausibility (a mechanism). We are further towards a causal association than before these studies were performed. However, we still have a lot of work to do before causality can be accepted.

The important point of our paper is that exposure to NSAIDs is associated with a severe adverse event, namely hospitalisation for incident (Crohn's and ulcerative) colitis. A milder, specific, NSAID colitis may exist. This is an attractive hypothesis which should be tested using epidemiological studies. We would be happy to collaborate with such a venture.

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1 Gleeson MH, Hardman JV, Clinton C. Colitis, non-steroidal anti-inflammatory drugs (NSAID's) & salicylates — a strong association [abstract]. *Gut* 1996;**39**(suppl 3):A243.

Surveillance of the duodenum in patients with familial adenomatous polyposis

EDITOR,—We compliment Vasen *et al* (*Gut* 1997;**40**:716–19) on their attempt to quantify the benefit of surveillance of the duodenum in patients with familial adenomatous polyposis (FAP). We should like to make the following points:

The St Marks's study[1] is not strictly comparable to the Scandinavian study in that in the former results are based on the use of a side-viewing endoscope to focus particularly on the high risk periampullary area, whereas the Scandinavian study used forward viewing endoscopes only.

Vasen *et al* state that pancreaticoduodenectomy has substantial morbidity and mortality; this statement contradicts another within the body of the text referring to the decline in mortality from this operation over the past decade. It would be fair to say that the operation has the potential for substantial morbidity and mortality. However, this potential has not been fulfilled, especially in the context of a performance of a pylorus-sparing pancreaticoduodenectomy (E Tiret and C Penna, Hôpital St Antoine, Paris, France, personal communication), together with encouraging results with regard to the lack of recurrence of duodenal adenomas.

We would suggest that a baseline screening examination be done before the age of 30. This might be done at the time of colectomy. This may be particularly worthwhile in those patients with a family history of duodenal cancer where such an examination would hopefully also reassure them.

There is additional evidence to that cited in favour of the existence of the adenoma–carcinoma sequence in the duodenum.[2] For example, adenomas were found in or adjacent to duodenal cancer in 84% (38/45) of patients with FAP.

We agree that the results of surveillance should be collected in a uniform manner at a central registry. In this way the quoted criteria for population screening, namely the natural history of duodenal ademonas, the

availability of curative treatment and evidence that this treatment leads to improved prognosis can be assessed. Without surveillance of these individuals, the natural history will remain unknown. Treatments will not be able to be instituted until symptoms, which are usually a marker of advanced disease, occur. The incentive for testing novel treatments, such as Sulindac and its derivatives, COX 2 agents, photodynamic laser therapy, antacid administration or gene therapy, would not be as great.

Finally, a point of personal sensitivity. Vasen *et al*'s reference to the "so-called" Spigelman classification neglects to say that this classification was "called-so" in the literature by the Scandinavian group itself![3] Whilst we remain in favour of eponymous labelling of conditions[4] and are indeed gratified to see that these authors have adopted the term Bussey–Gardner polyposis, the stimulus for labelling the classification of severity of upper gastrointestinal disease in FAP did not come from the St Mark's group.

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1 Spigelman AD, Williams CB, Talbot IC, *et al.* Upper gastrointestinal cancer in patients with Familial Adenomatous Polyposis. *Lancet* 1989;ii:783–5.
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4 Spigelman AD, Thomson JPS, Phillips RKS (eds). Nomenclature: familial adenomatous polyposis (Bussey–Gardner polyposis). In: *Familial adenomatous polyposis and other polyposis syndromes*. London: Edward Arnold, 1997:xiii–iv.

Reply

EDITOR,—We thank Drs Spigelman and Phillips for their remarks. We agree that the St Marks's study and the Scandinavian study are not strictly comparable. Between the two studies, however, there was little significant difference in the detection rate of stage IV duodenal polyposis.

We do not believe that our statements on the mortality associated with pancreaticoduodenectomy are contradictory, as we still consider a mortality of 5% to be substantial, especially if this surgical procedure is performed in a relatively young patient with a disease that is potentially malignant but still benign (stage IV duodenal polyposis). An important issue which was not mentioned in our manuscript is that recent studies indicate that mortality is significantly higher in hospitals with limited experience (fewer than five procedures per year).[1 2] We would therefore like to emphasise that such procedures should be performed at centres in which experience is available.

The disadvantage of recommending a baseline duodenoscopy before the age of 30 is that if abnormalities are found, most physicians will decide to continue (frequent) examinations, thereby imposing an additional burden on the patient. Although exceptions can be made for patients seeking reassurance, we prefer to make a decision on the basis of the natural history of the disease, which indicates that duodenal cancer before 30 years of age is extremely rare.

We regret that our comments on the Spigelman classification gave the impression that we dislike the name of this classfication. On the contrary, we believe that the classification proposed by Spigelman is very useful and fully deserves his name.

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1 Gordon TA, Burleyson GP, Tielsch JM, *et al.* The effects of regionalization on cost and outcome for one general high-risk surgical procedure. *Ann Surg* 1995;**221**:43–9.
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BOOK REVIEWS

Gastroesophageal Reflux Disease (GERD): Back to Surgery? Edited by Buchler MW, Frei E, Klaiber C, Krahenbuhl L. (Pp 248; illustrated: $184.00.) Basel: Karger, 1996. ISBN 3-8055-6476-7.

Gastro-oesophageal reflux disease (GORD) can now rightly be regarded as the English disease as its prevalence here exceeds that any of any other country.

This book is a response to the "swelling chorus of acclamation" relating to the place of minimally invasive surgery for the treatment of this condition and actually has a great deal to offer for anyone involved in the management of reflux patients.

In the 32 short essays there are some very important and highly relevant questions about GORD and an attempt to answer them. It is sometimes reassuring to know that despite a very informed discussion and sound reasoning, even the experts have uncertainties about, for example, Barrett's surveillance and how to manage the very exciting concept of the ultra-short Barrett's oesophagus.

The reappraisal of the diagnostic assessment of GORD is both timely and very helpful. Any attempt to abolish "equivocal oesophagitis" and to reduce the interobserver variation in this rather important and specific finding is, of course, welcome. There is no doubt that the major parts of the book are about when, how and who should do a "lapwrap". We are told that from the health economics point of view there is a break even point at six years when one compares a laparoscopic Nissen versus long term omeprazole. When one considers an open Nissen this break even point is not reached for 10 years. On the other hand in the USA, despite the existence of 40 000 Board Certified Surgeons there are probably only a small group of suitably trained surgical experts who can safely perform a laparoscopic fundoplication.

Several of the chapters debate the technical details of laparoscopic anti-reflux surgery and the surgeons will certainly welcome the discussions about the importance of mobilising short gastric vessels, the use of a Bougie, the length of the wrap as well as the place of a partial (Toupet) fundoplication. This book will also find a very welcome place with those who occasionally have to manage those unfortunate dysphagic patients after surgery and hopefully will approve the recommendation of a wait and see policy. A very sensible algorithm is presented for coping with this rather distressing complication. Perhaps an equally complexing problem are those with recurrent reflux after surgery, and it is particularly refreshing to have a frank discussion about the management of this group, emphasising the potential morbidity and even mortality of surgery in this particular scenario.

This book is about wrap versus zap. It is highly informative, at times entertaining, and will be of great value to those who are either in or hovering on the edge of the expanding field of laparoscopic anti-reflux surgery.

W J OWEN

Handbook of Anal Endosonography. Bartram CI, Frudinger A. (Pp 79; illustrated; £24.50.) Petersfield, UK: Wrightson Biomedical Publishing Ltd, 1997. ISBN 1 871816 35 1.

Since its introduction in 1989, anal ultrasound has revolutionised the assessment of anal sphincter injuries and incontinence, and sent shock waves through the obstetric and gynaecology community. It is now seen as an essential part of anorectal physiological testing and absolutely de rigueur prior to anal sphincter surgery. Colorectal surgeons, gastroenterologists, radiologists, and specialist nurses will welcome this excellent handbook.

Clive Bartram, the father of anal endosonography, with Andrea Frudinger, a former research fellow, has put together a series of high quality, mostly 10 MHz, images of the anal canal. The effects of patient position, age, sex, and site of the scanner within the anal canal are beautifully illustrated in the normal and abnormal anus including internal and external anal sphincter injuries, anal sepsis, and anal tumours.

This is a very practical book and an excellent guide to the interpretation of anal ultrasound images and a copy should be in every colorectal department or physiology laboratory in the country.

N J M MORTENSEN

The Liver and Systemic Disease. Gitlin N. (Pp 300; illustrated; £95.00.) Edinburgh: Churchill Livingstone, 1997. ISBN 0-443-05546-7.

All hepatologists and right-thinking physicians are well aware that the liver is not only the largest internal organ but also the most important. In systemic diseases, the liver is often affected and liver function disturbed; conversely, primary liver disease can affect every other organ system. While most of the major medical and hepatological textbooks have sections on the liver in systemic disease, this inevitably tends to be rather a rag-bag of topics. The laudable purpose of this book is "to provide a review of the hepatic manifestations occurring or resulting from diseases of other organs". To this end, Professor Gitlin has brought together a galaxy of eminent hepatologists from North America with a few non-hepatologists to produce a well presented and extensively illustrated book, and has largely succeeded in his aim.

There are 14 chapters which encompass the major body systems. Although this volume will be used as a reference source, there are several chapters which make compelling reading: those on the liver in diabetes and hyperlipidaemia, nutritional and haematological disorders, and sarcoidosis (which is rather a review of hepatic granulomas) were especially enjoyable to read. There are a few puzzling omissions: for example, I could find no mention in the index or within the appropriate chapters any discussion of the liver involvement in coeliac disease; iron overload is discussed briefly in the context of thalassaemia but no mention is made of haemochromatosis. There is only fleeting reference to the liver disorders associated with ulcerative colitis and Crohn's disease. With respect to the effect of liver disease on other organ systems, some areas, such as the hepatopulmonary syndrome, are fully covered, whereas there is no discussion on hepatorenal syndrome or hepatic osteopenia. There is scant discussion of the haemodynamic disturbances in either acute or chronic liver disease. Mechanisms of liver diseases are patchily covered: there is a full and clear description of the fatty liver yet the mechanism of sepsis related jaundice is only briefly discussed. Finally, it would have been helpful to have a review of postoperative and ITU jaundice.

This volume is a pleasure to look at and read; whether clinicians will wish to invest £95.00 for a book which provides little more information than may be found in major textbooks is less certain.

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Nutrition in Pediatrics—Basic Science and Clinical Application. 2nd edn. Edited by Walker WA, Watkins JB. (Pp 850; illustrated; price not given.) Hamilton: BC Decker Inc, 1996. ISBN 1-55009-026-7.

Readers of *Gut* might feel that paediatric nutrition is not among their main interests, but for those who seek to know what is happening in this closely allied field, through scientific curiosity or a desire to expand their clinical horizons, this book is a good starting point. Edited by two American paediatricians who have been energetic and influential in establishing paediatric gastroenterology as a distinct subspecialty, this book aims to do the same for paediatric nutrition. We believe that this book is of value both to trainees and those with an established interest in the field, and therefore we judge it from both viewpoints.

The novice will find that this book, divided into two halves, supports the editors' view that paediatric nutrition has come of age: general concepts, nutritional physiology and pathophysiology are well covered in the first half; and perinatal nutrition, nutritional aspects of specific disease states, followed by nutritional support, in the second. The balance between theory and practice reflects the sound basic research that has led to the growing importance of clinical nutrition to child health.

When it is estimated that more than three quarters of the world's children are undernourished, it is disappointing that the book is unashamedly directed at the developed world, with little on the aetiology, effects, recognition, and management of nutritional problems that occur in developing countries. Only half a paragraph is given to one of the most important advances in global child health in the past decade, the reduced mortality associated with reversal of mild vitamin A deficiency. The only reference to kwashiorkor is in the chapter on nutritional anaemias, and we could find little on protein-energy malnutrition in the developing world, even in the chapter devoted to malnutrition in hospitalised children. However, three chapters emphasise the importance of human milk and breast feeding to infant nutrition, and the amount of research that has gone into this field. It is encouraging to see the practical "Approach to breast-feeding" chapter included in a book otherwise largely concerned with science and disease.

The focus of textbooks has moved away from "nutrients" towards "nutritional support", recognising the critical part malnutrition plays in chronic childhood diseases. In considering specific diseases, the book recognises that the efficacy of nutritional support is proved in some areas (for example, Crohn's disease, short bowel syndrome, and renal failure), but avoids discussion of a more controversial and difficult topic, nutritional support in congenital heart disease. The final section includes chapters on parenteral and enteral nutrition.

For the specialist in paediatric nutrition this book compares well with its competitors, and in its second edition it is strong in clinical application. However, with its international authorship and presumed international readership it is a pity that there is not more on global problems in paediatric nutrition, which would broaden its appeal. Nevertheless, at a time when paediatric nutrition is close to standing alone as a distinct subspecialty, this book will find a secure place as a standard text for students, trainees, teachers, and practising paediatricians.

S C LING
L T WEAVER

NOTES

Course in Postgraduate Gastroenterology

A Course in Postgraduate Gastroenterology will be held in Oxford, UK, on 4–7 January 1998. This course has been designed for consultants and registrars, including those who do not specialise in gastroenterology. Topics will include:

- Liver disease
- Colonic neoplasia
- Acute pancreatitis
- Osteoporosis, arthritis and GI disease
- Food allergy and intolerance.

Course fee £200 ($330). Board and accommodation are available at Wadham College at extra cost. Six bursaries will be available for applicants training in gastroenterology or in research posts at British hospitals. Further information from: Dr DP Jewell, Gastroenterology Unit, Radcliffe Infirmary, Woodstock Road, Oxford OX2 6HE.

Colorectal Disease in 1998

The 9th Annual Colorectal Disease in 1998: An International Exchange of Medical and Surgical Concepts will be held at Marriott's Harbor Beach Resort, Fort Lauderdale, Florida, USA, from 19 to 21 February 1998. Further information from: Cleveland Clinic Florida, Department of Education, 2950 West Cypress Creek Road, Fort Lauderdale, FL 33309-1743, USA. Fax: 954 978 5539; Other: 800 359 5101, ext 5056; Local/international: 954 978 5056: email: jagels@cesmtp.ccf.org.

6th Southeast European Congress of Paediatric Surgery: Short Bowel Syndrome

The 6th Southeast European Congress of Paediatric Surgery: Short Bowel Syndrome will be held in Graz, Austria, on 22–23 May 1998. Further information from: Dr Günther Schimpl, Department of Paediatric Surgery, Auenbruggerplatz 34, A-8036 LKH-Graz, Austria. Tel: +43 316 385 3762; Fax: +43 316 385 3775.

9th British Association of Day Surgery Annual Scientific Meeting

The 9th British Association of Day Surgery Annual Scientific Meeting and Exhibition will be held at the Harrogate International Centre, Harrogate, UK, on 4–6 June 1998. Further information from: Kite Communications, The Silk Mill House, 196 Huddersfield Road, Meltham, West Yorkshire HD7 3AP, UK. Tel: 01484 854575; Fax: 01484 854 576; email: info@kitecomms.co.uk.

9th International Symposium on Cells of the Hepatic Sinusoid

The 9th International Symposium on Cells of the Hepatic Sinusoid will be held in Christchurch, New Zealand, from 27 September to 1 October 1998. Further information from: Professor Robin Fraser, I.S.C.H.S., Christchurch School of Medicine, PO Box 4345, Christchurch 8001, New Zealand. Tel: +64 3 3640 587; Fax: +64 3 3640 593; email: grogers@chmeds.ac.nz.

Growth Factors and Nutrients in Intestinal Health and Disease

An International Symposium on Growth Factors and Nutrients in Intestinal Health and Disease will be held at the Rihga Royal Hotel, Osaka, Japan, from 31 October to 3 November 1998. Further information from: Kinya Sando, MD, Department of Pediatric Surgery, Osaka University Medical School, 2-2 Yamadoaka, Suita, Osaka 565, Japan. Tel: +81 6 879 3753; Fax: +81 6 879 3759; email: gut@pedsurg.med.osaka-u.ac.jp.

Advanced Course in Gastroenterology

An Advanced Course in Gastroenterology will be held at the Royal College of Physicians of Edinburgh, UK, from 3 to 7 November 1998. Further information from: Miss Lee Ross, Symposium Assistant, Education, Audit and Research Department, Royal College of Physicians of Edinburgh, 9 Queen Street, Edinburgh EH2 1JQ, UK. Tel: +44 131 225 7324; Fax: +44 131 220 4393.